REMARKS

These remarks are in response to the office action mailed July 26, 2005. Claims 1-14 are currently pending. Claims 1-8 and 14 are rejected and claims 9-13 are withdrawn from consideration. Claim 1 is amended herein. Support for the amendment can be found throughout the specification. For example, support for the amendment to claim 1 can be found on page 3, lines 18-19. No new matter is believed to have been introduced.

REJECTION UNDER 35 U.S.C. §103

Claims 1-8 and 14 stand rejected under 35 U.S.C. § 103 as purportedly being unpatentable over Bush et al., American Journal of Physiology – Renal Physiology, 277:211-218 (1999) combined with U.S. Patent 6,015,659 to Welch et al. Applicants respectfully traverse this rejection.

The Examiner asserts that Bush et al. teaches that pretreatment of kidney epithelial cell cultures with tunicamycin prior to ATP depletion resulted in increased expression of the endoplasmic reticulum (ER) molecular chaperones and cytoprotection, thus allegedly teaching that pretreating cells with agents that induce endoplasmic reticulum molecular chaperones results in cytoprotection in the face of ATP depletion.

The Examiner further asserts that Welch et al. teach that benzoquinonoid ansamycins and specifically geldanamycin may be administered to cells that may be expected to experience a stress in order to stimulate production of Hsp proteins and therefore induce tolerance in the cells of the organism. Therefore, according to the Examiner, it would have been obvious to one of ordinary skill in the art at the time the invention was made to administer both tunicamycin and geldanamycin to epithelial cells in order to improve the cells' response to ischemia with the expectation that the cells so treated will benefit from the additive effects of the two different pathways of protection.

Applicants respectfully submit that combination of these disclosures fail to teach or suggest the claimed invention.

The claimed invention is directed at modulating the deterioration of cells as a result of ischemic injury. Specifically, the invention comprise four separate mechanisms: (a) inhibiting internalization of one or more intercellular junction proteins; (b) promoting activation of specific signaling events during short-term ischemia; (c) inhibiting degradation of proteins necessary for the maintenance of the polarized epithelial cell phenotype; and (d) enhancing protein folding and assembly capacity in the ER and/or cytosol. See for example claim 1. The cited references do not disclose the claimed combination of cellular strategies to combat ischemic injury in a multifaceted approach to repair this cellular process. Clearly, there is no suggestion to put (a), (b), and (c) together as claimed in the subject application.

Applicants submit that there is no motivation to combine the references to arrive at Applicant's invention. Thus, in view of the above, Applicants respectfully request the withdrawal of the rejection.

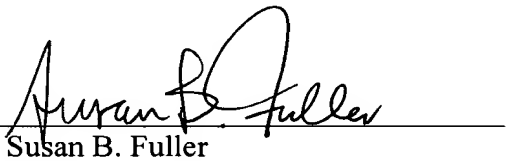
Conclusion

For the reasons set forth above, it is believed that this case is in condition for allowance. Applicants accordingly request that this Amendment be entered and that the rejection under 35 U.S.C. § 103 be carefully reconsidered. In the event that there are any questions relating to this application, it would be appreciated if the Examiner would telephone the undersigned concerning such questions so that the prosecution of this application may be expedited.

Respectfully submitted,

BUCHANAN INGERSOLL LLP

Date: October 18, 2005

By: 

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